Vaginal Cytological Findings during Pregnancy in the Mothers of Three Anencephalic Babies

Case No.	E.I.	K.I.	Vaginal Flora	Clumping	Other Features
1	Just above normal	High	Variable	Variable	Occasional parabasal cells
2	Just above normal	High	Variable	Moderate to good	Occasional parabasal cells
3	Variable; mainly normal	Variable but high (up to 40%) in 3rd trimester	Variable	Variable	Nil

E.I. = eosinophilic index
K.I. = karyopyknotic index

was having difficulties in accepting the diagnosis, and karyotyping of mother and baby was deferred. MacNaughton[8] reviewed the evidence that mothers of such babies may in the non-pregnant state have an abnormal steroid metabolism. An increased output of dehydroepiandrosterone has been reported in young mothers of mongoloid children[9 10] but this has been questioned by others[11] who discount the possibility that the differences present are due to translocations being present in only some of the mothers while others have a normal karyotype. However, the opportunity to study the hormonal status during pregnancy in such a condition does not often arise, and further studies may well indicate that some of the gene loci involved in regulating oestrogen metabolism during pregnancy reside in the affected chromosomes.

The presence of non-specific, unexplained, persistently abnormal hormonal cytological patterns in pregnancy in groups at risk may alert the clinician to the possibility of certain congenital defects being present and indicate the necessity for further investigations.

I would like to acknowledge the encouragement of Professors W. I. C. Morris and F. A. Langley and a research grant from the United Manchester Hospital.

—I am, etc.,

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Can I Have an Ambulance, Doctor?

SIR,—The article by Dr. T. C. Beer and others (9 February, p. 226) provides some very disturbing facts, not so much about the use of the ambulance service but about physiotherapy. What it reveals is that a treatment centre attached to one of the most modern hospitals in the country provides such a low level of treatment that the author's comments about the effectiveness of outpatient physiotherapy are almost bound to be correct. The last sentence is really the important one: "Doctors ordering [physiotherapy] should remember that it may be merely a potentially socially disruptive placebo."

The facts revealed in the article showed that 75% of the patients treated were receiving no more than one hour of physiotherapy per week. I find it incredible, at a time when rehabilitation services are being reassessed and recommendations made about the implementation of the Tunbridge Committee report and when consultants in rehabilitation are being appointed, that such a very low level of treatment with a modern rehabilitation centre which treats patients on a full-time basis—that is, the patients attend for a 37-hour working week and treatment is devoted to active exercise or effective forms of physiotherapy and recovery time is reduced to an absolute minimum thereby.

I wonder if this article is in fact illustrating a norm for patient after-care because, if it is, no wonder the rehabilitation services are in such urgent need of reappraisal and reorganization.—I am, etc.,

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Cot Deaths

SIR,—With reference to your very informative leading article (2 March, p. 341) the following factors seem *always* to be present when cot deaths occur: (1) horizontal position; (2) bed rest; (3) quiet sleep for some hours; (4) the baby is clothed and the covers are wrapped round the child; (5) a helpless and somewhat weak child; (6) an unsuspected and silent event; and (7) a greater or lesser degree of interstitial oedema in the lungs.[1]

The most reasonable pathogenesis for cot deaths according to these premises is, I think, that the baby is drowned in its own body fluid. This body fluid is more abundant in newborn babies and highly mobile. Interstitial oedema in the lungs hampers oxygen transport through the respiratory membrane, especially in the hypostatic parts of the lungs. The hypoxaemia which follows produces drowsiness, insensibility, and death.

If this theory is right the fatal event should never occur when: (*a*) the child is out of bed; (*b*) the baby is crying, when the oedema would be driven out of the chest; (*c*) the child is naked—heavy covers in the winter season, constricting clothes, and bulky nappies under the buttocks would tend to drive more blood into the thoracic cavity; (*d*) the child sleeps with the upper half of the body elevated; (*e*) the child has strength enough to get out of the flat, recumbent position; and (*f*) there has been insufficient time since washing and feeding for enough oedematous fluid to gather.

In 1969 I was telephoned by a distressed mother who had just found her 31-day-old infant motionless and apparently dead. I immediately ordered the child to be placed in a vertical position. When I saw her some 6-7 minutes later breathing was present but hardly audible. The whole body was very pale, with some traces of scum around the mouth. The head and the limbs were quite flaccid. In the next 30 minutes recovery was very rapid. The baby became normal, yawned healthily, and wanted food.[2] The girl has been in good health since then but somehow weaker than the two older children, who were twins.

Other potentially fatal diseases in which fluctuations in body fluid may be of decisive importance are croup[3-5] (acute laryngitis) and pulmonary oedema due to heart failure. At necropsy the disastrous oedema may well be gone—as is the rule in dead bodies—into the common pool of hypostatic fluid.—I am, etc.,

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High-dose Frusemide in Renal Failure

SIR,—With reference to the paper by Professor F. Cantarovich and others (24 November 1973, p. 449) and the subsequent letter from Dr. D. Ganeval and his colleagues (9 February, p. 244) I should like to report my experience of the use of high-dose frusemide in the treatment of acute tubular necrosis and exacerbations of chronic renal failure.

In my series 10 patients were treated with frusemide, initially 1g intravenously and rising gradually to 3g over a period of seven days if no response was obtained; a control group of 10 patients were treated conventionally, without frusemide. The patients were allocated to the two groups at random and were evenly matched for age and diagnosis.

I found that in the group treated with frusemide the period of oliguria was shorter and the diuresis was greater than in the controls, so fluid retention was not a problem; there was no life-threatening hyperkalaemia and the patients were discharged earlier (mean seven days). The most practical factor was that in the control group seven patients required peritoneal dialysis, while in the group receiving high-dose frusemide only one patient required peritoneal dialysis.—I am, etc.,

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SIR,—I have read with interest the paper by Professor F. Cantarovich and others (24

November, p. 449). In this department we have used frusemide successfully in the treatment of 13 cases of acute renal failure in which the oliguria was of more than 48 hours duration.[1] Failure was post-obstetric in 10 cases, post-surgical in one, traumatic in one, and due to myocardial infarction in one. The doses used were modest compared with those used by Professor Cantarovich and his colleagues, except in one case in which a total dose of 6g was given in addition to dialysis. No side effects have been noticed.

We strongly feel that frusemide is safe and should be given priority in a country like Pakistan where economic limitations may not allow the use of more sophisticated method of treatment. The drug should be used early to avoid irreversible renal damage. This, we feel, is a new role of frusemide on a prophylactic basis.—I am, etc.,

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1 Siddique, Y. K., Jafarey, S. N., and Naqvi, A. J., *Journal of the Pakistan Medical Association*, 1974, **24**, 62.

Hypertension and Myocardial Infarction

SIR,—In your leading article (5 January, p. 1) entitled "Presentation of Myocardial Infarction" you stated that hypertension predisposed to this complication. Though hypertension may coexist with coronary artery disease, all the evidence tends to suggest that this is a defence mechanism and that a high aortic diastolic pressure is essential to force blood through the narrowed, inelastic coronary vessels in sufficient quantity to prevent infarction.

Friedberg and Horn[1] showed that myocardial necrosis followed functional coronary insufficiency in patients suffering from acute haemorrhage, and Scherf and Klotz[2] demonstrated electrocardiographic changes resembling those of myocardial infarction in 14 out of 15 patients suffering from gastrointestinal haemorrhage. These are uncommon causes of myocardial infarction but they are the only circumstances in which it has been shown conclusively that general medical disasters have precipitated myocardial infarction. In these conditions the blood pressure is low. In 200 cases of sudden death associated with coronary heart disease investigated by the coroner in Perth, Western Australia, in the past nine months only 12 of the victims were engaged in pursuits which could have caused a temporary rise in blood pressure at the time of onset of symptoms. The remaining 188 were resting, sleeping, moving slowly, or recovering after exercise. Whether the term myocardial infarction is used in its clinical sense to denote a syndrome which can cause death (which was present in all cases) or whether it is restricted to those cases exhibiting macroscopic evidence of infarction (42, including two who were exercising) hypertension, systolic or diastolic, played no consistent role in producing these infarctions. They seemed to arise when the circumstances suggested hypotension rather than hypertension.

This contrasts with a random selection of 12 patients suffering from coronary artery disease and angina of effort subjected to angiography and discussed at the weekly cardiology conferences at the Royal Perth Hospital. These patients had averaged well over 500 anginal attacks each in the previous six months. These attacks were associated with effort or emotion (and presumably increased systolic and diastolic blood pressure) but none seemed to have developed acute infarction from this hypertension. The attacks responded to rapidly acting nitrites (pyridinolcarbamate; Anginin) which reduced the blood pressure. There is, on the other hand, no evidence that a myocardial infarct or the myocardial infarction syndrome has ever improved on similar treatment.

Do you think you might modify your view that hypertension is a causal or predisposing factor in myocardial infarction?—I am, etc.,

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Care of the Coronary Patient

SIR,—Dr. K. Astrad and his colleagues (23 March, p. 567) found no significant change in mortality from acute myocardial infarction after the establishment of a coronary care unit in their hospital. I should, however, like to emphasize the fact that their unit is not representative of the majority of coronary care units, from which indeed much lower mortality figures are quoted. Though their unit possesses electronic monitoring equipment it lacks the most important feature a coronary care unit should possess and that is a high staff-to-patient ratio with a staff which is fully trained and *specialized* in the prompt identification and management of cardiac arrhythmias. The staff in their unit is relatively small and is also responsible for the admission and 24-hour care of *all* medical emergencies at a time when they must require maximum attention. One also gets the impression of their unit bustling with activity at all times (about 5,000 admissions a year) and one wonders what effect this atmosphere has on the coronary patient, who is liable to develop arrhythmias at the least adrenergic provocation. It is clear that the conclusions drawn from their paper will not necessarily apply to the more common, relatively small, self-contained coronary care unit.

I was also very interested in the tentative conclusions drawn by Dr. A. Colling in his well-reasoned paper on the subject of home or hospital care for the coronary patient (23 March, p. 559). I would, however, question the wisdom of keeping the patient at home any longer than absolutely necessary if he is seen within two hours of onset of the pain unless a defibrillator is available. Admittedly by monitoring the cardiac rhythm and giving appropriate therapy as required it is possible to minimize the chances of the patient developing ventricular fibrillation, but this dangerous arrhythmia often develops with little or no warning and in spite of prior antiarrhythmic therapy. After all, if ventricular fibrillation occurs at home and does not respond to a vigorous thump on the chest and a short period of cardiac massage only urgent defibrillation may save the patient, so the patient should be sent to the vicinity of a defibrillator as soon as possible just in case this is urgently needed. Monitoring of the patient's rhythm and appropriate treatment can be continued in the ambulance. Secondly, if a patient is seen within 24 hours but more than two hours after onset of the symptoms I feel the bias should be towards hospital care. Arrhythmias are still quite common during this time and the patients' life may well depend on their prompt detection and management. Adequate analgesia and reassurance should always precede the patient's journey to hospital.—I am, etc.,

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Working Hours of Junior Staff

SIR,—A great deal has been written recently about the implementation on 1 July of the agreement to reduce the existing standard working week for junior hospital staff from 102 to 80 hours. N.H.S. authorities and hospital medical staff have been asked to try to devise rota schemes to secure for junior staff the increase in off-duty time which is, and has always been, the object, but all concerned with negotiation or implementation have realized throughout that this objective could not be achieved everywhere at once. As there are so many doubts being expressed it seemed timely to my colleagues on the Staff Side of the Joint Negotiating Committee for Hospital Medical and Dental Staff that I should, as chairman of the J.N.C. attempt to clarify the position.

The profession came to the conclusion nine years ago that "it is not only quite unreasonable, but against the best interests of the patients, that a young doctor should ever be on call for 168 hours in a week, and on active duty for 80 hours or more. . . . The young doctor must be allowed a reasonable amount of off-duty for rest, recreation and study." That quotation is taken from a review of terms and conditions published by the then Central Consultants and Specialists Committee in 1965.[1] The object has been restated repeatedly since then, and the present proposal—a clinical commitment not normally to exceed 80 hours—was approved by the Central Committee for Hospital Medical Services[2] and presented in its annual report to the National Conference of Representatives of Hospital Medical Staff in 1972.

With this well-established policy on record, an agreement in principle to implement on 1 July 1974 was made between the representatives of the profession and of the Health Departments in March 1973—to allow adequate time for preparation. The first results of some pilot studies—at King's College Hospital, London, and at Derby and Norwich—confirm our expectation that the one-in-three rota (with no duty over 80 hours) is possible at present in some hospitals and divisions within hospitals but not in others. This was always foreseen, and until such time as either the staffing position can be improved or agreed arrangements can be made for more effective use